

# Genetic Structure of the *Bovicola ovis* (Mallophaga: Trichodectidae) in Southwestern Australia

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**ABSTRACT** The sheep biting louse, *Bovicola ovis* (Schränk), is an economically important, world-wide ectoparasite of sheep. In Australia, up to 30% of sheep flocks are infested with lice. The usual method of control has been synthetic pyrethroids applied as pour-on along the back of the sheep, but treatment failures have become widespread since 1985 because of the development of resistance. We used allozyme markers to study the distribution of genetic variation within and among populations of lice on different farms in Western Australia. Genetic variation within populations was similar to previously reported values for other ectoparasitic arthropods. Heterozygote deficiencies were found at 1 locus in a number of population and another 2 loci in 1 other population. However, another variable locus conformed to Hardy–Weinberg equilibrium and there was little evidence of extensive linkage disequilibrium between loci. Further studies are necessary to establish the breeding system. Genetic differences among populations were not related to geographic separation, which is consistent with an island model of population structure. A small but significant proportion (2.8%) of the total genetic variation was distributed among populations' equivalent under the island model to a gene flow of 8.7 individuals exchanged per generation. The implications of this result are discussed in terms of controlling and managing synthetic pyrethroid resistance in sheep biting lice.

**KEY WORDS** sheep lice, population genetics, gene flow, pyrethrin resistance

THE RATE OF development of resistance to insecticides depends on the mode of inheritance of resistance, the initial frequencies of alleles conferring resistance or susceptibility, the relative fitness of the corresponding genotypes, the selection pressure imposed by insecticide usage and the amount of gene flow among populations (Roush and McKenzie 1987). Any critical assessment of the various options for managing insecticide resistance requires empirical estimates of these parameters. Gene flow or genetically effective migration between populations of pests or parasites is important because it determines the rate of spread of resistance genes into populations that are currently susceptible, and because it determines the rate of spread of susceptible genes into populations where resistance is developing. Which of these processes is most important depends on the relative frequencies of resistant and susceptible alleles, and the level of gene flow. Simulation studies have suggested that when resistance alleles are rare, high levels of gene flow will increase the rate of evolution of resistance by spreading resistance alleles among subpopulations (Caprio and Tabashnik 1992a). Conversely, when resistance alleles are widespread, the provision of untreated refuges that permit the survival of susceptible genotypes, or the release of susceptible genotypes into resistant populations, can greatly slow the evolution of resis-

tance (Taylor and Georgiou 1979, Alstad and Andow 1995). Although there seems to be a consensus that gene flow is one of the most important influences on the management of resistance, little is known about its magnitude for most pest species (Roush and Daly 1990, Denholm and Rowland 1992, McCaughey and Whalon 1992).

The sheep biting louse, *Bovicola ovis* (Schränk), is an economically important ectoparasite found throughout most sheep-raising areas of the world. In Australia, 10–30% of sheep flocks are infested with lice (Levot 1995). Heavy infestations reduce wool production and fiber quality. Synthetic pyrethroid (SP) insecticides, applied as a pour-on along the back of the sheep, have been a popular method of control in Australia since 1981. Treatment failures, as a result of the development of SP resistance, were first reported in 1985 and resistance is now widespread (Johnson et al. 1990, Levot and Hughes 1990, Levot 1995). In Western Australia, SP resistance occurs throughout the state, but there is a large variance in resistance among farms, as measured by *in vitro* assay. This may be caused by either restricted gene flow between populations of lice on different farms or by local variation in selection pressure that maintains differences among populations in the face of gene flow (Caprio and Tabashnik 1992b).

Ecological evidence suggests that gene flow may be restricted in *B. ovis*. Sheep biting lice are very small (up to 1.8 mm long) and flightless, and they spend

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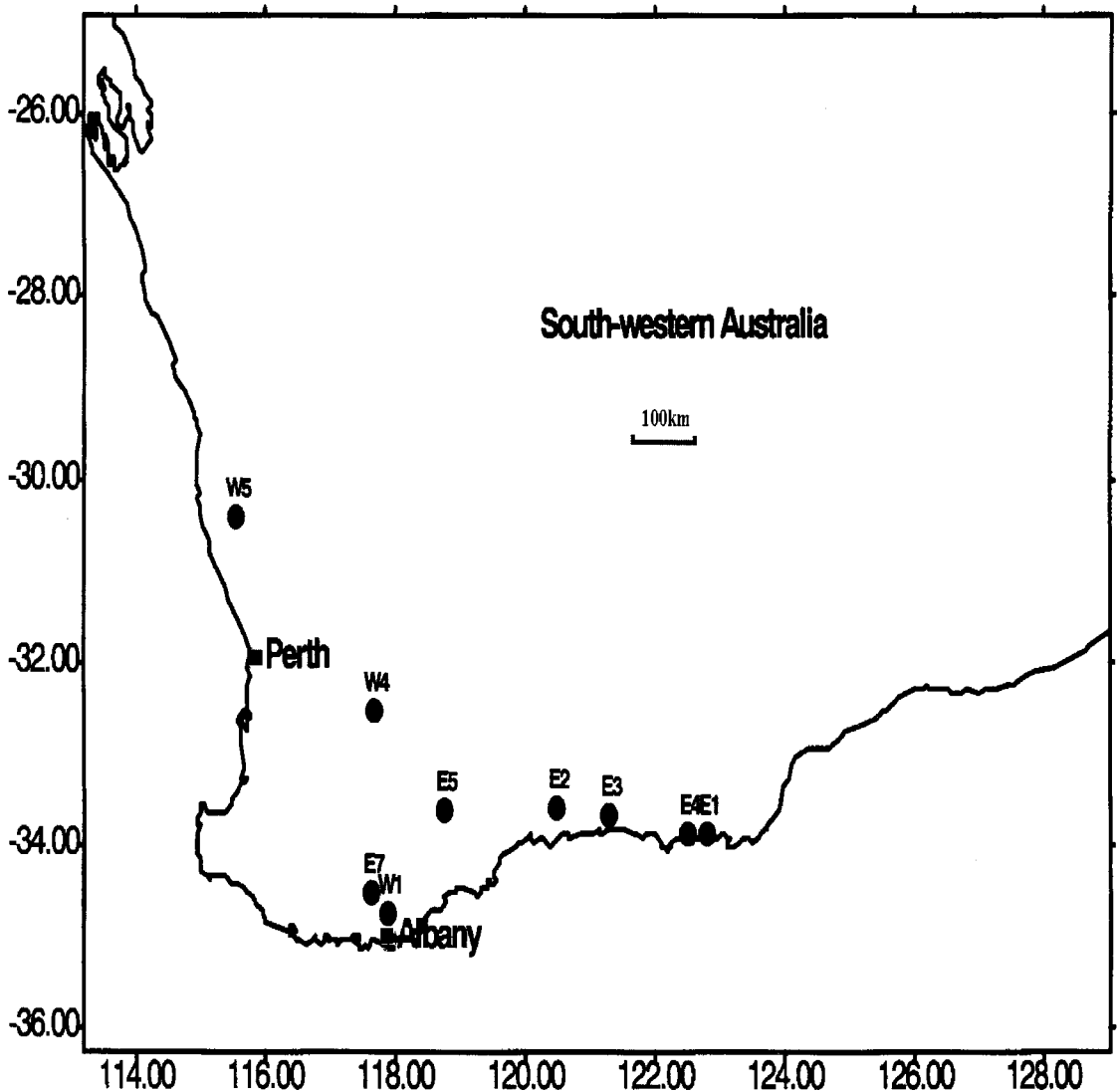


Fig. 1. Localities from which populations of *B. ovis* were sampled.

their entire lives on the host. Transmission of the parasite is assumed to occur only through direct sheep to sheep contact, which limits migration between populations (Arundel 1988, Levot 1995). The aim of this study was to estimate gene flow among populations of lice on different farms in Western Australia by measuring genetic variation within and among populations using allozyme markers.

#### Materials and Methods

**Samples.** Between 30 and 190 (mean 66), lice were sampled from 2 to 6 sheep on each of 9 farms in the southwest of Western Australia (Fig. 1). The sample size of 190 from population E1 was used to check for the presence of alleles occurring at low frequency; such alleles were found for 2 peptidase loci. Prelimi-

nary studies from population E1 found no genetic differences among lice from different sheep on the same farm, and this level of population substructure was not considered in this study. All lice were frozen at  $-85^{\circ}\text{C}$  until required for electrophoresis.

**Electrophoresis.** Individual lice were placed in Eppendorf tubes and homogenized in 20  $\mu\text{l}$  of grinding buffer. After centrifuging for 3 min at  $1,300 \times g$ , the supernatant was extracted and loaded onto cellulose acetate gels (Helena Laboratories, Helena, Australia). All gels were electrophoresed in tris citrate (pH 7.8) or tris glycine (pH 8.5) buffer, with a current of 2 mA per gel applied for 20–25 min at  $4^{\circ}\text{C}$ . Gels were stained for specific enzyme activity following the methods of Richardson et al. (1986) and Hebert and Beaton (1989). Enzyme banding patterns were interpreted genetically using standard, conservative criteria

(Richardson et al. 1986). Six enzymes were examined in all individual lice: glucose phosphate isomerase (encoded by the locus *Gpi*), malate dehydrogenase (*Mdh*), malic enzyme (*Me*), leucylglycylglycine peptidase (*Pep A*), leucyl tyrosine peptidase (*Pep B*), and leucyl proline peptidase (*Pep C*). At each locus, alleles are designated by letter, beginning with the allele responsible for the most anodally migrating allozyme.

**Analysis.** Genetic diversity within populations was described by the proportion of loci at which the frequency of the most common allele was <99% (*P*), the mean number of alleles per locus (*A*), and the mean expected unbiased heterozygosity or total gene diversity (*H*; Nei 1978), with the variance in heterozygosity calculated by the method of Nei (1987). For polymorphic loci, genotypic frequencies expected under Hardy-Weinberg equilibrium were calculated from allelic frequencies using Levene's (1949) correction for small sample size. Deviations of observed from expected frequencies were tested by chi-square. Where >2 alleles were present at a locus, all the least common alleles were pooled before analysis. The extent of deviation from Hardy-Weinberg equilibrium within populations was expressed by Wright's fixation index (*F*), with the approximate variance of Brown (1970). Across all populations, *F* values were summarized by the weighted mean, *F*<sub>IS</sub>, calculated by the method of Nei and Chesser (1983). The departure of *F*<sub>IS</sub> from 0 was tested by chi-square (Workman and Niswander 1970). Burrow's composite measure of linkage disequilibrium ( $\Delta_{AB}$ ) was estimated for pairwise combinations of variable loci and tested for significance by chi-square as outlined by Weir (1990).

Genetic differentiation among populations was described by *F*<sub>ST</sub>, the standardized variance of allelic frequencies, and was calculated by the method of Nei and Chesser (1983). The departure of *F*<sub>ST</sub> from 0 was tested by chi-square (Workman and Niswander 1970). Gene flow among populations was estimated as *N<sub>e</sub>m* = (1/*F*<sub>ST</sub>-1)/4, where *N<sub>e</sub>* is the effective population size and *m* is the migration rate (Wright 1951). Isolation by distance among populations was tested by regressing log-transformed estimates of gene flow against log-transformed geographic distances between each pair of populations, as suggested by Slatkin (1993). The significance of the relationship between gene flow and geographic distance was assessed by Mantel's test, implemented using the resampling procedure of Manly (1985).

The measures described above were calculated by the computer programs GENESTRUT (Constantine et al. 1994) and LD86.FOR (Weir 1990). Where multiple statistical tests of the same hypothesis were performed, significance levels were adjusted by the Bonferroni procedure to a group-wide type I error rate of 5%.

## Results

**Genetic Variation within Populations.** Over all populations, 4 of the 6 loci assayed were polymorphic, although 2 of the loci were variable in only 1 popu-

**Table 1.** Allelic frequencies at polymorphic loci in samples of *B. ovis* from 9 populations

Locus	Allele	Population								
		E1	E2	E3	E4	E5	E7	W1	W4	W5
<i>Gpi</i>	a	0.01	0.00	0.06	0.00	0.05	0.04	0.03	0.00	0.02
	b	0.97	1.00	0.91	1.00	0.93	0.96	0.97	1.00	0.98
	c	0.02	0.00	0.03	0.00	0.02	0.00	0.00	0.00	0.00
<i>Mdh</i>	a	0.06	0.00	0.11	0.04	0.00	0.00	0.00	0.00	0.00
	b	0.88	0.93	0.89	0.87	1.00	1.00	0.94	1.00	1.00
	c	0.06	0.07	0.00	0.09	0.00	0.00	0.06	0.00	0.00
<i>Pep A</i>	a	0.99	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	b	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
<i>Pep B</i>	a	0.99	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	b	0.01	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

lation (E1). Allelic frequencies for each population are shown in Table 1 and standard measures of genetic diversity in Table 2. Population W4 was monomorphic for all loci, but all other populations were polymorphic for at least 1 locus. Over all populations, *P* = 0.24, *A* = 1.33, and *H* = 0.027.

Significant deficits of heterozygous genotypes were found for *Mdh* in all populations at which the locus was polymorphic, and for *Pep A* and *Pep B* in the 1 population at which these loci were polymorphic. By contrast, genotypic frequencies for *Gpi* conformed to Hardy-Weinberg equilibrium in all populations where the locus was polymorphic (Table 3). Over all loci, *F*<sub>IS</sub> was significantly >0 (Table 3).

**Linkage Disequilibrium.** Associations between *Gpi* and *Mdh* were tested in the 3 populations where both loci were variable: E1, E3, and W1. In only 1 of these populations (E1), was significant linkage disequilibrium detected ( $\Delta_{AB}$  = 0.023 ± 0.013, *P* < 0.05).

**Genetic Variation among Populations.** There were significant differences in allelic frequencies among populations at *Gpi* (*F*<sub>ST</sub> = 0.016, *P* < 0.05) and *Mdh* (*F*<sub>ST</sub> = 0.036, *P* < 0.05). Over all loci, *F*<sub>ST</sub> = 0.028 (*P* < 0.05) (i.e., 2.8% of the total genetic variance) was caused by differences among populations equivalent under the island model of population structure to a gene flow of 8.7 migrants exchanged per generation.

The plot of pairwise gene flow estimates against geographic distance showed no spatial pattern to genetic variation among populations (Fig. 2). Geo-

**Table 2.** Proportion of polymorphic loci (*P*), mean number of alleles per locus (*A*) and total gene diversity (*H*) in samples of *B. ovis* from 9 populations

Population	<i>P</i>	<i>A</i>	<i>H</i>
E1	0.67	2.00 (0.80)	0.054 (0.034)
E2	0.17	1.17 (0.17)	0.023 (0.023)
E3	0.33	1.50 (0.70)	0.061 (0.039)
E4	0.17	1.33 (0.67)	0.039 (0.039)
E5	0.17	1.33 (0.67)	0.022 (0.022)
E7	0.17	1.17 (0.17)	0.013 (0.013)
W1	0.33	1.33 (0.27)	0.029 (0.020)
W4	0.00	1.00	0.00
W5	0.17	1.17 (0.17)	0.005 (0.005)
Mean	0.24	1.33	0.027

Variances in parentheses.

Table 3. Heterozygosities observed ( $H_o$ ) and expected ( $H_e$ ) under Hardy–Weinberg equilibrium, and fixation indices ( $F$ , with variances in parentheses) at polymorphic loci within populations of *B. ovis*

Locus		Population								F <sub>IS</sub>	
		E1	E2	E3	E4	E5	E7	W1	W4		W5
Gpi	H <sub>o</sub>	0.056	—	0.071	—	0.138	0.083	0.061	—	0.029	-0.020
	H <sub>e</sub>	0.066	—	0.162	—	0.131	0.081	0.060	—	0.029	
	F	0.140	—	-0.078	—	-0.050	-0.033	-0.021	—	0.000	
		(0.022)		(0.006)		(0.003)	(0.006)	(0.007)		(0.000)	
Mdh	H <sub>o</sub>	0.005	0.000	0.000	0.000	—	—	0.000	—	—	0.994*
	H <sub>e</sub>	0.217	0.137	0.205	0.237	—	—	0.116	—	—	
	F	0.975*	1.000*	1.000*	1.000*	—	—	1.000*	—	—	
		(0.001)									
Pep A	H <sub>o</sub>	0.000	—	—	—	—	—	—	—	—	1.000*
	H <sub>e</sub>	0.021	—	—	—	—	—	—	—	—	
	F	1.000*	—	—	—	—	—	—	—	—	
Pep B	H <sub>o</sub>	0.011	—	—	—	—	—	—	—	—	0.502*
	H <sub>e</sub>	0.021	—	—	—	—	—	—	—	—	
	F	0.466*	—	—	—	—	—	—	—	—	
		(0.095)									
Mean											0.628*

$F_{IS}$  is the weighted mean of  $F$  values across populations. —, Monomorphic loci.  
\*, Significant at the 5% level, with the Bonferroni correction.

graphic distance explained only 1% of the variation in pairwise gene flow estimates ( $g = -2.3, P > 0.5$ ).

Discussion

There have been few previous studies on the genetic structure of lice, and none on *B. ovis*. Genetic variation within populations of *B. ovis* was low when compared with mean values for internal parasites ( $P = 0.31 \pm 0.03, H = 0.08 \pm 0.01$  for 38 species; Lymbery 1995) or free living invertebrates ( $P = 0.38 \pm 0.01, H =$

$0.10 \pm 0.01$  for 371 species; Nevo et al. 1984), but was similar to that reported in other studies on ectoparasitic arthropods ( $P = 0.14 \pm 0.01, H = 0.04 \pm 0.01$  for 10 species; Bull et al. 1984; Hilburn and Sattler 1986; Sattler et al. 1986; Nadler and Hafner 1989; Barker et al. 1991). In addition, *B. ovis* has colonized Australia relatively recently (with the introduction of sheep in the late 18th century), and colonizing populations are often less genetically diverse than their source populations (Selander and Kaufman 1973, Hillis et al. 1991).

Heterozygote deficiencies were found at 3 loci, with a mean fixation index of 0.628. This is unlikely to be a consequence of unobserved population substructuring (Wahlund effect), because we found no evidence of genetic differences between lice from different sheep on the same farm and heterozygote deficiencies were maintained when lice from single sheep were analyzed as separate populations (unpublished data). Inbreeding or selection may be responsible for the observed heterozygote deficiencies. These causes are usually distinguished by the consistency of genotypic frequency deviations over loci, as the breeding system will affect all loci equally, but selection is locus-specific. Inbreeding therefore seems a possible explanation, because 3 out of 4 loci showed significant heterozygote deficiencies. However, 2 of these loci were polymorphic at the 1% level in only 1 population. Linkage disequilibrium is a typical concomitant of inbreeding (Allard et al. 1968), but is difficult to detect when allelic frequencies are very asymmetrical or sample sizes are small. We found linkage disequilibrium between the 2 most variable loci in only 1 out of 3 populations. Further studies using more variable loci are need to determine whether *B. ovis* populations are inbred.

We found small, although significant, genetic differentiation among populations of lice from different farms at the 2 most variable enzyme loci. There was no

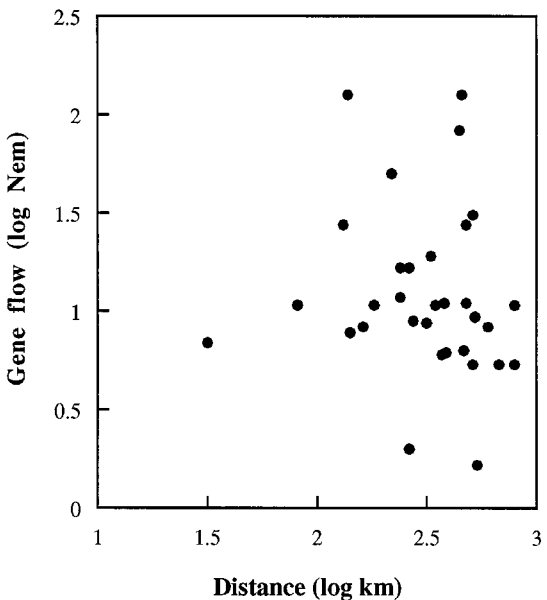


Fig. 2. Regression of log-transformed pairwise gene flow estimates ( $Nm$ ) and log-transformed pairwise geographic distances between populations of *B. ovis* in Western Australia.

evidence that genetic differences between populations were related to geographic separation (isolation by distance). This may indicate that drift/migration equilibrium has not been reached because of recent colonization (Slatkin 1993), or may reflect an island model of population structure, where all populations are equally accessible for gene flow (Wright 1931). Although the island model is usually considered an idealized population structure, it may in fact be characteristic of many parasites of domestic livestock, whose movements are dictated by human economic activity (Blouin et al. 1995).

Gene flow among populations of *B. ovis* was estimated at 8.7 individuals exchanged per generation. This value was calculated from the estimated  $F_{ST}$  over loci of 0.028, and is therefore dependent on the assumptions of the island model of population structure, including negligible selection and mutation operating on the loci, and an equilibrium between gene flow and genetic drift. The gene flow estimate should be treated with caution, because it is based on only 4 loci, and only 2 of these loci were polymorphic in >1 population. To have more confidence in the reality of the assumptions upon which the estimate is based, we need to examine a larger number of loci. Given the limited genetic variation within populations of lice, the most efficient way of collecting the required data would be through the application of DNA techniques, such as microsatellite markers. Microsatellite studies would also enable the application of newer analytical techniques that consider genealogical relationships among genotypes, providing a more effective separation of demographic processes that influence population structure from the genetic processes responsible for historical branching events (Bossart and Prowell 1998).

Despite these caveats, the current data do suggest that gene flow among lice populations in Western Australia is great enough to rule out genetic drift as a significant evolutionary force. Under the island model, a gene flow >1 will prevent the genetic differentiation (fixation of alternative alleles) of populations through genetic drift, although allelic frequency differences may still arise (Allendorf and Phelps 1981). Genetic differentiation may still occur through selection, as long as the selection coefficient ( $s$ ) is greater than the rate of migration ( $m$ ) among populations (Haldane 1930).

What are the implications of these results for the control of SP resistance in *B. ovis*? First, the estimated rate of gene flow is great enough to rapidly spread resistance alleles among populations. The variance in SP resistance among farms in Western Australia cannot be caused solely by restricted gene flow, but must also be an effect of differences in selection pressure, caused by differences in farm management. This provides, therefore, an empirical demonstration of the effectiveness of management techniques in slowing the evolution of insecticide resistance. Second, the estimated rate of gene flow is not great enough to delay the development of resistance through the reverse migration of susceptibility alleles. Computer

simulations by Caprio and Tabashnik (1992a) showed that resistance evolution was not delayed by the migration of susceptibility alleles into resistant populations unless the migration rate ( $m$ ) was >10%. With a gene flow ( $N_e m$ ) of 8.7 individuals per generation,  $m > 10\%$  would imply an effective population size ( $N_e$ ) of <80 individuals. Although there are no estimates of effective population size in *B. ovis*, ecological data indicate that it is likely to be at least an order of magnitude greater than this (Arundel 1988), meaning that the migration rate must be much less than 10%. This implies that some artificial enhancement of migration, such as the release of susceptible individuals into resistant populations, would be required to effectively reverse resistance development.

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